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Virulence, Aggressiveness, Evolution, and Distribution of Races of *Puccinia striiformis* (the Cause of Stripe Rust of Wheat) in North America, 1968-87

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Abstract

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Thirty-nine races of *Puccinia striiformis*, the fungus that causes stripe rust of wheat, have been identified in North America, beginning with four races present when monitoring began in 1968. Additional races were detected as follows: one each in 1968, 1972, 1975, 1978, 1980, and 1982; two each in 1974, 1984, and 1985; three each in 1981 and 1987; five in 1977; and six each in 1976 and 1983. Eleven of the races were first detected on newly released wheat cultivars that were resistant to previously known races. Those 11 races caused severe epidemics within 3 years after release of the cultivars. Within a few years after detection of the new virulences, new combinations of virulences appeared. Reassortment of all existing virulences occurred in the absence of a sexual cycle.

North America can be divided into seven wheat-growing regions of stripe rust occurrence, based on geographic barriers, prevailing winds and other weather, cropping methods, and virulence of the races. They are region 1: eastern Washington and Oregon, northern Idaho, and eastern British Columbia; region 2: western Montana and southern Alberta; region 3: southern Idaho and northern Utah; region 4: western Oregon and northern California; region 5: northwestern Washington and western British Columbia; region 6: central California; and region 7: the area east of the Rocky Mountains.

In region 1, 31 races of *P. striiformis* have been detected, of which 26 were first detected in the region. The other five races probably evolved independently in region 1 after they were detected in other regions. Races in regions 2 and 3 were detected either later in the same year that severe epidemics of the races occurred in region 1 or within a few years, suggesting that races in regions 2 and 3 originated in region 1. Most new virulences were associated with the release of cultivars with high race-specific resistance, but some virulences in regions 5 and 6 were not associated with resistance of cultivars grown in the regions. Other factors, such as wild hosts and the presence of indigenous races, appear to contribute to the diversity of races in regions 5 and 6. Region 5 has 18 of the 39 races, of which 6 are found in only that region. Region 6 has 11 races, of which 2 are unique for that region. One race, which is virulent on Lemhi but not on other differential cultivars, occurs in only regions 5 and 6.

Three races detected in California have a narrow range of virulence on the differential wheat cultivars and lack aggressiveness. Races that are virulent on the cultivar Chinese 166 (gene Yr_1) were less aggressive than races virulent on Heines VII (gene Yr_2) and Fielder (gene Yr_6). Races virulent on Chinese 166 are less prevalent even in plots that have been inoculated with the races, and they develop more slowly and produce fewer spores.

Races virulent on the differential cultivars Lemhi and Heines VII were detected in all regions; on Druchamp, Produra, Stephens, and Fielder in regions 1, 2, 4, 5, and 6; on Yamhill in regions 1, 4, 5, and 6; on Lee in regions 1, 4, and 5; on Moro in regions 1, 2, 4, and 5; and on Paha (Suwon 92/Omar) and Tyee in only region 1. Races virulent on *Triticum spelta album*, Compair, and Riebesel 47-51 have not been detected in North

America. Genes for resistance in the differential cultivars can be postulated, based on the virulence of the races on those cultivars.

Keywords: *Puccinia striiformis*, stripe rust of wheat, yellow rust of wheat, race-specific resistance, forecasting, monitoring, disease control, epidemiology, differential wheat cultivar

Contents

Virulence, Aggressiveness, Evolution, and Distribution of Races of *Puccinia striiformis* (the Cause of Stripe Rust of Wheat) in North America, 1968-87

Roland F. Line and Abdul Qayoum

Introduction

Stripe rust, caused by *Puccinia striiformis* West., is the most important disease of wheat in western North America, and it has become more important in the South-Central United States since 1984. The disease was first recognized in North America in 1915 by F. Kolpin Ravn, a visiting scientist from Denmark, who was traveling in the Western United States with a U.S. Department of Agriculture crop survey team (Carleton 1915). Once recognized, the disease was detected throughout the Western United States in 1915 and in Canada within a few years (Humphrey et al. 1924, Johnson and Newton 1928). Later, H.B. Humphrey (Humphrey et al. 1924), after examining herbarium specimens, determined that *P. striiformis* had been present in western Washington for at least 23 years before it was recognized. Early plant pathologists were aware of the destructiveness of stripe rust in Europe and Asia and were concerned about its possible spread to the major wheat-growing regions in the United States and

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central Canada (Hungerford 1923, Humphrey et al. 1924, Newton and Johnson 1936). Consequently, several major studies were conducted during the 1920's. However, the only reports on the pathogenicity of *P. striiformis* in North America were on its host range and on the resistance of local cultivars. Hungerford and Owens (1923) reported that *Bromus sterilis* was resistant to stripe rust collections from *Hordeum jubatum* but not to those from *Elymus glaucus* and *Bromus marginatus*. That report indicated the existence of races, or strains, of *P. striiformis*.

Newton et al. in 1932 (Newton et al. 1933) and Bever in 1933 (Bever 1934) each isolated two physiologic forms (races) of *P. striiformis* using differential wheat cultivars. Newton and Johnson (Newton and Johnson 1936) reported that their two races were similar to the race from Montana and the race from Idaho reported by Bever. The race from Montana was virulent on Chinese 166 and Red Russian and avirulent on Heines Kolben. The collection from Moscow, Idaho, was virulent on Heines Kolben and avirulent on the other two cultivars.

No reports were published on the virulence of *P. striiformis* in North America for the next 25 years. Concern about stripe rust diminished, the disease was no longer considered important in North America, and research projects on stripe rust were eliminated. But the situation changed drastically in the late 1950's and early 1960's, when severe epidemics caused massive losses in California and the Pacific Northwest (Shaner and Powelson 1971, Tollenaar and Houston 1967). These devastating effects of stripe rust led to an increased emphasis on breeding for resistance to the rust. Purdy and Allan (Purdy and Allan 1963) observed that at least three stripe rust races existed in Washington. Additional races were subsequently identified in the Western United States (Purdy and Allan 1966; Beaver and Powelson 1969; Line 1972, 1976, 1980, 1984; Volin and Sharp 1973).

The races detected in the early 1930's were identified on a set of differential cultivars that had been used in Europe (Gassner and Straib 1932). But that set did not effectively differentiate the predominant races of *P. striiformis* in North America. Therefore, a set of differential cultivars for the United States was selected in 1969, and a uniform system of describing and naming the races was agreed on by those working on stripe rust in the United States (Line et al. 1970). Since then, the system, with some modification, has been used to monitor the virulence and distribution of pathotypes (races) in North America. Additional differential cultivars were added as new races appeared (Line 1972, 1976, 1980). This report summarizes the results of more than 20 years of research on the differentiation, virulence, and aggressiveness of races of *P. striiformis* in North America; detection of the races; geographic distribution of the races; interrelationships of the races; and movement of inoculum from region to region.

Field Monitoring

To monitor the prevalence and severity of stripe rust and to detect the changes in virulence of the pathogen, trap plots of winter wheat and/or spring wheat were planted annually from 1968 to 1987 in commercial fields or at experiment stations. The plots consisted of single 1.5- to 4-m rows of past and current commercial cultivars, new improved lines of wheat, and cultivars and lines that differentiate races of *P. striiformis* in North America and Europe. Initially, all of the monitoring sites were in the Pacific Northwest, but as the need became evident, sites were added at other locations in the Western United States where stripe rust occurred. In 1986 and 1987, additional monitoring sites were established in Colorado and at several locations in the South-Central United States. Each year, the trap plots were planted at 15-25 sites. The winter wheat plots were planted in fall; the spring wheat plots were planted in fall in central California and in spring at other sites. Wheat cultivars and lines in national uniform disease nurseries, international rust nurseries, regional and breeding nurseries, and demonstration plots were also used to provide supplemental data on rust. The plots were visited one to five times during the growing season; on each visit, rust intensity (severity), infection type (table 1, fig. 1), and stage of plant growth were recorded for each cultivar or line in the plots. Plots at sites within 600 km (375 miles) of Pullman, WA, were most frequently examined. When visits to more distant sites were not possible, some disease data were recorded by cooperators. When traveling to and between the sites, we made frequent stops to examine commercial fields and recorded data on rust severity, infection type, and stage of growth for those fields.

Collecting and Evaluating Rust Samples for Virulence

Each year, 100-300 rust samples collected from wheat lines or cultivars in the monitoring plots and commercial fields and samples sent in by cooperators were evaluated on seedlings of differential wheat cultivars under controlled temperatures using the technique described by Line et al. (Line et al. 1970). Some samples received from cooperators were not viable. Seven differential cultivars were initially used (Line et al. 1970). Later, other differential cultivars were added when races appeared that could not be differentiated by the older differentials. Then both the older rust collections and the new collections were evaluated on both the older and new differentials. The differentials and the year that they were added are listed in table 2.

Table 1. Scale for recording stripe rust (*Puccinia striiformis* West.) infection types

Infection type	Signs and symptoms for infection types[1]
0	No visible signs or symptom
1	Necrotic and/or chlorotic flecks; no sporulation
2	Necrotic and/or chlorotic blotches or stripes; no sporulation
3	Necrotic and/or chlorotic blotches or stripes; trace sporulation
4	Necrotic and/or chlorotic blotches or stripes; light sporulation
5	Necrotic and/or chlorotic blotches or stripes; intermediate sporulation
6	Necrotic and/or chlorotic blotches or stripes; moderate sporulation
7	Necrotic and/or chlorotic blotches or stripes; abundant sporulation
8	Chlorosis behind sporulating area; abundant sporulation
9	No necrosis or chlorosis; abundant sporulation

[1]Blotches occur on seedlings, and stripes occur on plants in later stages of growth.

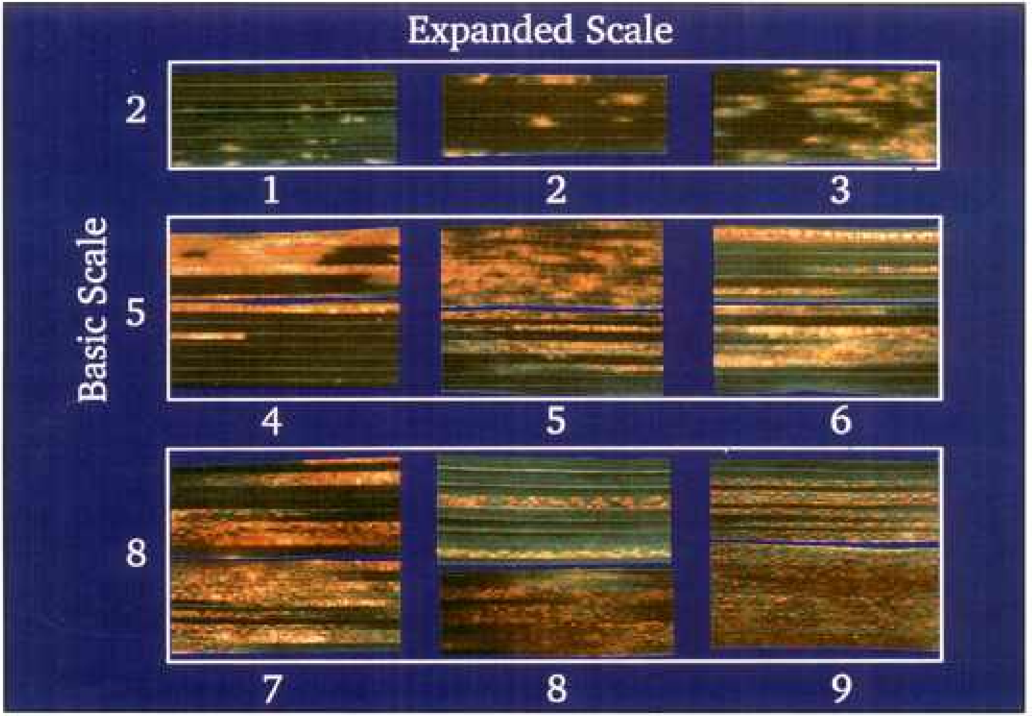

Figure 1. Basic scale and expanded scale for recording infection types of *Puccinia striiformis* on adult wheat plants.

Table 2. Differential wheat cultivars used to identify races of *Puccinia striiformis* in North America

Differential		Cultivar		Year added to differential set
Number	Name	Number	Type	
1	Lemhi	CI011415	Spring	1968
2	Chinese 166	CI011765	Winter	1968
3	Heines VII	PI201195	Winter	1968
4	Moro	CI013740	Winter	1968
5	Suwon 92/Omar[1]	CI013749	Winter	1968
5	Paha	CI014485	Winter	1974
6	Druchamp	CI013723	Winter	1969
7	Riebesel 47-51	PI295999	Winter	1969
8	Produra	CI017406	Spring	1974
9	Yamhill	CI014563	Winter	1974
10	Stephens	CI017596	Winter	1976
11	Lee	CI012488	Spring	1977
12	Fielder	CI017268	Spring	1980
13	Tyee	CI017773	Winter	1983

[1]Suwon 92/Omar (CI013749) was replaced by Paha (CI014485) in 1974 because it is grown in Northwestern United States, has the same parentage, and differentiates the same resistance as CI013749.

Seeds of the wheats used to maintain cultures, increase inoculum, and identify races were planted in a potting mixture of 24 L peat, 8 L perlite, 12 L sand, 12 L Palouse silt loam, 16 L vermiculite, 350 g lime, 250 g 14-14-14 Osmacote, and 150 g ammonium nitrate. The seedlings were grown before and after inoculation under fluorescent lights (from 1968 to 1976) or metal halide lights (from 1977 to 1987) to supplement natural daylight and to extend the photoperiod to 16 hr. Seedlings in the early two-leaf stage were inoculated with spores and placed in a dew chamber at 10°C for 12-15 hr. Then the seedlings were placed in a greenhouse at temperatures programmed to change gradually between a minimum of 2°-5°C at 0200 hours during the 8-hour dark period and a maximum of 18°-20°C at 1400 hours during the 16-hour light period.

If the collection consisted of only a few rusted leaves, the inoculum was increased on seedlings of highly susceptible cultivars (Michigan Amber, *Triticum spelta saharensis*, Nugaines, or Lemhi) or on seedlings of the same cultivar from which the sample was collected. Then spores from these plants were used to inoculate the differential cultivars. Tests on the differential cultivars were usually repeated one or more times to confirm the results, especially if the pattern of virulence was unusual. If two or more infection types were evident on a differential cultivar (an indication of a mixture of races), subcultures from that cultivar as well as other differential cultivars were increased on that cultivar and on cultivars that showed no mixture, and each subculture was retested on the differential cultivars. This technique for separating races was sometimes repeated several times. If the races were not adequately separated by that method, spores from individual pustules were transferred to rust-free plants within 1 day after the first evidence of sporulation, and inoculum from those single pustules was retested on the differential cultivars. Spores of each collection and subculture were stored in liquid nitrogen, usually before evaluation of the collections and subcultures, for use in reevaluating questionable results.

Data on infection types, based on a 0-9 scale (table 1, fig. 2) were recorded once at 15-18 days after inoculation and one or more times at 21-35 days after inoculation. Infection types were determined on both first and second seedling leaves. If the infection types on the first and second seedling leaves differed, the infection type on the second leaf (if different, the infection type on the second leaf was always higher) was used as the infection type to represent that seedling. The two or more sets of data on infection types were summarized and converted to avirulence/virulence data. The pathogen was considered avirulent when there were no symptoms or there were necrotic or chlorotic flecks, necrotic or chlorotic blotches without sporulation, or necrotic or chlorotic blotches with only a trace of to slight sporulation (infection types 0, 1, 2, 3, and 4). The pathogen was considered to be virulent if it resulted in moderate to abundant sporulation, with or without chlorosis or necrosis (infection types 5, 6, 7, 8, and 9).

When a new race of *P. striiformis* was identified, single-pustule isolates and, in some cases, single-spore isolates were selected from one of the cultures, increased, and retested on the differential cultivars. Once identified, one culture was selected as the type race and designated by a Cereal Disease Laboratory (CDL) number. (CDL refers to the U.S. Department of Agriculture Cereal Disease Research Laboratory at Pullman, WA.)

Differentiation of Races

The method of classifying and describing races and the rules for naming races proposed by Line et al. (Line et al. 1970) were used. The seven cultivars of wheat that differentiated races in 1969 are represented by the numbers 1 to 7. A slash (/)

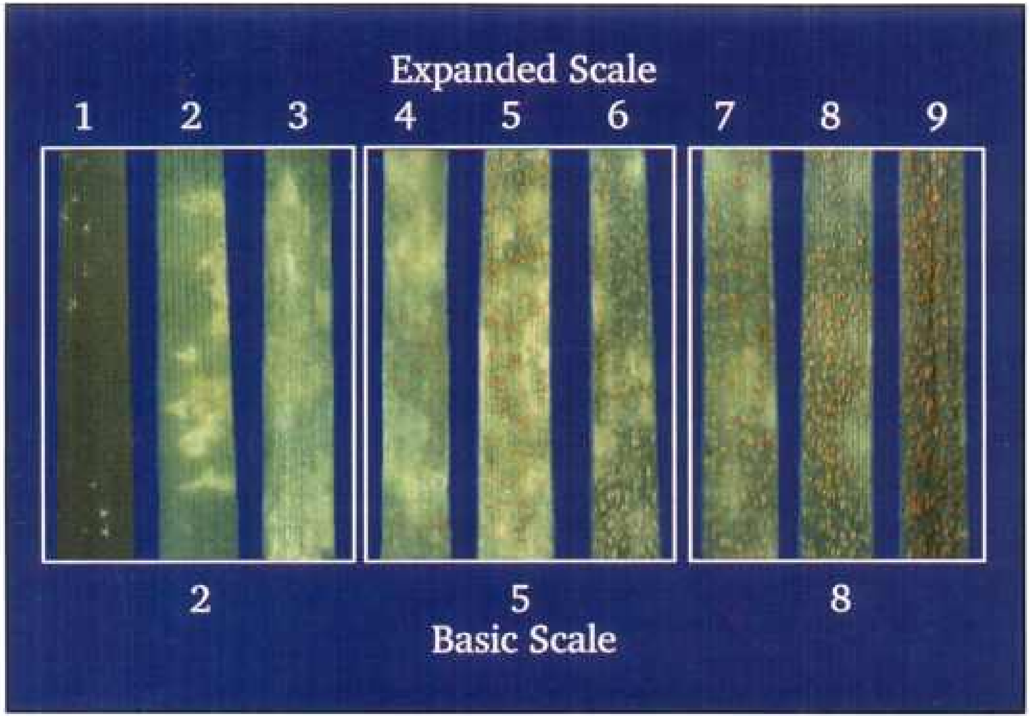

Figure 2. Basic scale and expanded scale for recording *Puccinia striiformis* infection types on wheat seedlings.

separates differentials on which the pathogen is avirulent from differentials on which the pathogen is virulent. The description 2,4,5,6,7/1,3 indicates that the race is avirulent on differentials 2, 4, 5, 6, and 7 and virulent on differentials 1 and 3. Differentials can be added or deleted whenever more information on the pathogenicity of *P. striiformis* becomes available. Six differential cultivars have been added since the first seven differentials were proposed. Thus the system of identifying and differentiating the races of *P. striiformis* in North America has evolved along with changes in the pathogen. Without the addition of new differentials, most of the new races would appear to be the same as the older races, but knowledge of the virulence of the new races is important in developing resistant cultivars and in managing resistance.

The description of races provides continuity from year to year and among researchers and regions. As long as the races are described, their names are less important. The races can be designated by any name that an individual researcher wishes to use. When the proposal on classifying, describing, and naming races was published in 1970 (Line et al. 1970), workers in Montana, Oregon, and Washington were identifying races of *P. striiformis* in the United States. Since then, all race monitoring in North America has been conducted in the senior author's laboratory. Once identified and described, the races were sequentially designated by a CDL number. CDL-1 is the first race that was identified and CDL-39 is the last (table 3, fig. 3). Each CDL race is a type race, i.e., the race on which the specific avirulence/virulence pattern was first described. For instance, race CDL-3 is the type race described as 2,4,5,6,7,8,9,10,11,12,13/1,3 (table 3). Theoretically, each collection could be a uniquely different biotype, and other collections with the same avirulence/virulence pattern as the type race could differ if enough differentials are used. However, all collections with the same

Table 3. Virulence and avirulence of North American races of *Puccinia striiformis*, type race designation, and total number of races virulent on each differential cultivar

Description[1] of North American race	CDL[2] type race	Differential wheat cultivar[3]												
		1	2	3	4	5	6	7	8	9	10	11	12	13
3,4,5,6,7,8,9,10,11,12,13/1,2	1	V[4]	V	A[4]	A	A	A	A	A	A	A	A	A	A
3,4,6,7,8,9,10,11,12,13/1,2,5	2	V	V	A	A	V	A	A	A	A	A	A	A	A
2,4,5,6,7,8,9,10,11,12,13/1,3	3	V	A	V	A	A	A	A	A	A	A	A	A	A
2,4,5,6,7,11,12/1,3	4	V	A	V	A	A	A	A				A		A
2,5,6,7,8,9,10,11,12,13/1,3,4	5	V	A	V	V	A	A	A	A	A	A	A	A	A
2,3,4,5,7,9,10,11,13/1,6,8,12	6	V	A	A	A	A	V	A	V	A	A	A	V	A
2,4,6,7,8,9,10,11,12,13/1,3,5	7	V	A	V	A	V	A	A	A	A	A	A	A	A
2,4,5,6,7,8,10,11,12,13/1,3,9	8	V	A	V	A	A	A	A	A	V	A	A	A	A
2,4,5,7,9,10,11,13/1,3,6,8,12	9	V	A	V	A	A	V	A	V	A	A	A	V	A
4,5,6,7,8,10,11,12,13/1,2,3,9	10	V	V	V	A	A	A	A	A	V	A	A	A	A
2,3,4,5,6,7,8,9,10,11,12,13/1	11	V	A	A	A	A	A	A	A	A	A	A	A	A
2,3,4,7,8,9,10,11,13/1,5,6,12	12	V	A	A	A	V	V	A	A	A	A	A	V	A
2,3,4,7,9,10,11,13/1,5,6,8,12	13	V	A	A	A	V	V	A	V	A	A	A	V	A
2,3,4,5,6,7,9,10,11,13/1,8,12	14	V	A	A	A	A	A	A	V	A	A	A	V	A
2,4,5,7,8,9,11,12,13/1,3,6,10	15	V	A	V[5]	A	A	V	A	A	A	V	A	A	A
2,4,5,6,7,8,10,12,13/1,3,9,11	16	V	A	V	A	A	A	A	A	V	A	V	A	A
4,5,6,7,8,10,12,13/1,2,3,9,11	17	V	V	V	A	A	A	A	A	V	A	V	A	A
2,5,6,7,8,10,11,12,13/1,3,4,9	18	V	A	V	V	A	A	A	A	V	A	A	A	A
2,4,5,7,9,11,13/1,3,6,8,10,12	19	V	A	V	A	A	V	A	V	A	V	A	V	A
2,3,4,5,7,9,11,13/1,6,8,10,12	20	V	A	A	A	A	V	A	V	A	V	A	V	A
1,3,4,5,6,7,8,9,10,11,12,13/2	21	A	V	A	A	A	A	A	A	A	A	A	A	A
2,4,5,6,7,8,9,10,11,13/1,3,12	22	V	A	V	A	A	A	A	A	A	A	A	V	A
2,4,5,7,8,11,12,13/1,3,6,9,10	23	V	A	V[5]	A	A	V	A	A	V	V	A	A	A

Table 3—Continued

Description[1] of North American race	CDL[2] type race	Differential wheat cultivar[3] 1	2	3	4	5	6	7	8	9	10	11	12	13
2,4,6,7,8,9,10,11,13/1,3,5,12	24	V	A	V	A	V	A	A	A	A	A	A	V	A
2,4,5,7,11,13/1,3,6,8,9,10,12	25	V	A	V	A	A	V	A	V	V	V	A	V	A
2,4,5,6,7,8,10,11,13/1,3,9,12	26	V	A	V	A	A	A	A	A	V	A	A	V	A
2,4,5,6,7,8,9,10,11/1,3,12,13	27	V	A	V	A	A	A	A	A	A	A	A	V	V
2,5,6,7,8,9,10,11,13/1,3,4,12	28	V	A	V	V	A	A	A	A	A	A	A	V	A
2,6,7,8,9,10,11,12,13/1,3,4,5	29	V	A	V	V	V	A	A	A	A	A	A	A	A
2,3,5,7,9,10,11,13/1,4,6,8,12	30	V	A	A	V	A	V	A	V	A	A	A	V	A
2,4,6,7,8,9,10,12,13/1,3,5,11	31	V	A	V	A	V	A	A	A	A	A	V	A	A
2,3,5,6,7,8,9,10,11,12,13/1,4	32	V	A	A	V	A	A	A	A	A	A	A	A	A
2,4,5,6,7,8,10,11/1,3,9,12,13	33	V	A	V	A	A	A	A	A	V	A	A	V	V
2,6,7,8,9,10,11,13/1,3,4,5,12	34	V	A	V	V	V	A	A	A	A	A	A	V	A
2,3,4,5,6,7,8,9,11,12,13/1,10	35	V	A	A	A	A	A	A	A	A	V	A	A	A
2,5,6,7,8,10,11,13/1,3,4,9,12	36	V	A	V	V	A	A	A	A	V	A	A	V	A
2,4,5,7,13/1,3,6,8,9,10,11,12	37	V	A	V	A	A	V	A	V	V	V	V	V	A
2,4,5,6,7,8,9,10,12,13/1,3,11	38	V	A	V	A	A	A	A	A	A	A	V	A	A
3,5,6,7,8,9,10,11,12,13/1,2,4	39	V	V	A	V	A	A	A	A	A	A	A	A	A
Total virulent races		38	6	26	9	8	11	0	9	11	7	5	18	2

[1] As described by Line at al. See text on Differentiation of Races.
[2] Cereal Disease Laboratory (CDL) type race.
[3] 1 = Lemhi, 2 = Chinese 166, 3 = Heines VII, 4 = Moro, 5 = Paha, 6 = Druchamp, 7 = Riebesel 47-51, 8 = Produra, 9 = Yamhill, 10 = Stephens, 11 = Lee, 12 = Fielder, 13 = Tyee.
[4] V = virulent, A = avirulent.
[5] Initially avirulent (infection type = 2), but eventually virulent (infection type = 6-7).

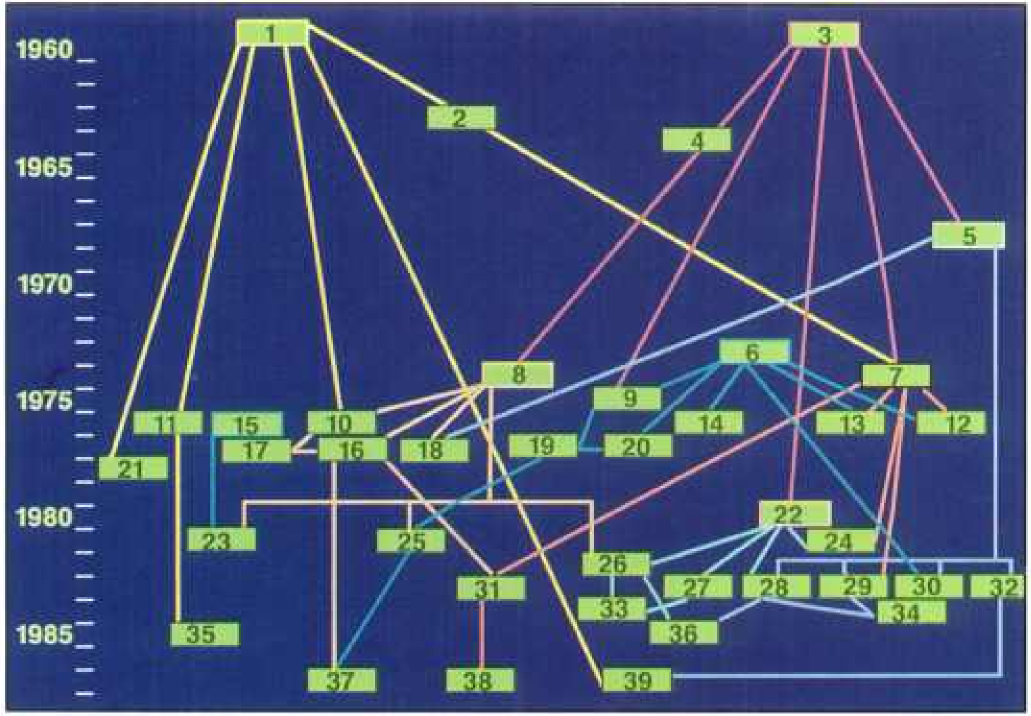

Figure 3. Chronological appearance of new races of *Puccinia striiformis* in North America from 1960 to 1987, their interrelationships based on common virulences, and their possible evolution.

avirulence/virulence pattern are referred to in this discussion by the same CDL number; for example, all races with the avirulence/virulence description 2,4,5,6,7,8,9,10,11,12,13/1,3 are referred to as CDL-3.

The descriptive and classification system for avirulence/virulence is easy to use and provides the essential information about the virulence of all races. In a binary system proposed by Johnson et al. (Johnson et al. 1972), differential cultivars 1, 2, 3, 4, 5, 6, 7, and 8 are assigned decanary values 1, 2, 4, 8, 16, 32, 64, and 128, respectively, and the sum of the decanary values for the cultivar(s) on which the race is virulent is used as the race number (race name). Thus, race 197 would be a race that is virulent on differential cultivars 1, 3, 7, and 8 (1 + 4 + 64 + 128). That system is more cumbersome. For example, a race that is virulent on only differential 13 would be designated as race 4096, and if a race is virulent on only differential 15, it would be race 16,384. If the race is virulent on several cultivars, the races are even more difficult to sort out. A race virulent on cultivars 1, 3, 13, and 15 (1 + 4 + 4096 + 16,384) would be designated as race 20,485. Also, the system discourages including new differentials. Only one new differential cultivar has been added since the system was first proposed in 1972 (Stubbs 1985), even though new races are known to exist that can be differentiated only by the use of additional cultivars.

Discovery and Occurrence of Races in North America

Table 3 contains the following information on the 39 races of *P. striiformis* that have been detected in North America since 1960: the virulence of the races on the 13 differential wheat cultivars currently used to identify the races, the CDL type-race designation (North American races), and the avirulence/virulence description of the races. Figure 3 shows the chronological appearance of the races, their interrelationships based on the virulence patterns in table 3, and the possible evolution of the races. Figure 4 depicts the seven regions of North America where the races have been detected.

Four races (CDL-1, CDL-2, CDL-3, and CDL-4) were identified before 1968. From the early 1960's to the late 1970's, CDL-1 was used to inoculate research plots near Pullman, WA (region 1); Corvallis, OR (region 4); and Bozeman, MT (region 2). During that period, CDL-1 was never detected more than 40 km (25 miles) from the inoculation sites. The race was detected in two regions where it was not used to inoculate plots: western Washington (region 5) in 1976 and California (region 6) in 1977. CDL-1 has never been detected in regions 3 or 7, and it remains extremely rare in all other regions.

CDL-2 (like CDL-1 but virulent on Suwon 92/Omar, CI013749) was first collected in eastern Washington in 1963 (Purdy and Allan 1963). CDL-2 was reported to occur in other regions (Purdy and Allan 1966), but we have found it in only region 1, where it is extremely rare. We did not collect isolates with the virulence of CDL-2 until 1978, after detection of other races that are virulent on CI013749.

CDL-3 was the predominant race from 1961 to 1974 in regions 1, 2, 3, and 4 and in the early 1970's in region 6. It may have been present in western Washington (region 5) and east of the Rocky Mountains (region 7), but those regions were not surveyed during that period. CDL-3 is frequently collected in region 5 and is currently the predominant race in region 7. It is the only race that commonly occurs throughout North America.

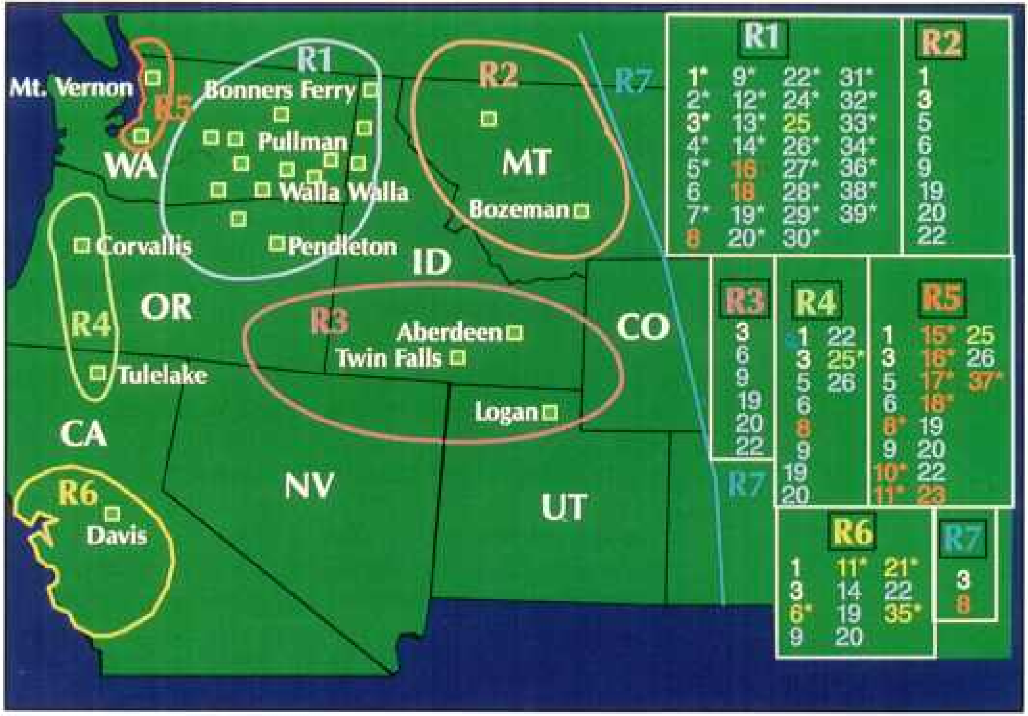

Figure 4. Regions of stripe rust occurrence in North America and lists of races of *Puccinia striiformis* that occurred in each region. R1 is region 1, R2 is region 2, etc. Asterisks and color indicate races first detected in each region. Symbol □ in map shows location of sites for monitoring stripe rust in western North America (sites in Colorado and South-Central United States are not shown).

The single isolate of CDL-4 differed from CDL-3 on the wheat cultivar Alba. CDL-4 was highly virulent on Alba (infection type 8-9), whereas CDL-3 initially produced a low infection type (type 2) on Alba; by about 1 week later, the infection type had increased to type 7. No additional information about CDL-4 is available because the culture was lost.

CDL-5 is similar to CDL-3, but it is also virulent on Moro and cultivars that have the major gene for stripe rust resistance from the plant introduction line PI178383. CDL-5 first appeared in 1968 near Bonners Ferry in northern Idaho (region 1), where it reduced the yield of Moro by more than 30%. By 1969, CDL-5 had spread to eastern Montana (region 2), where it severely damaged Crest, which also has resistance from PI178383. CDL-5 continued to occur in eastern Washington and northern Idaho but did not immediately spread to central Washington, where Moro was grown extensively and is still grown. CDL-5 was used in the early 1970's to inoculate research plots in region 4. The race was never detected more than 40 km (25 miles) from the inoculation site. In 1977, CDL-5 appeared for the first time in region 5 along with CDL-18, which is also virulent on Moro. Since 1978, neither race has been detected in region 5, probably because cultivars with resistance from PI178383 are not grown in region 5. In the early 1980's, the prevalence of CDL-5 and other races virulent on Moro increased in central Washington, especially on the cultivar Jacmar, which was even more susceptible than Moro. Moro is still grown in the drier parts of central Washington because of its superior emergence, and the races CDL-5 and CDL-18 continue to occur throughout region 1.

CDL-6 first appeared in 1973 on Pitic 62 near Davis, CA (region 6). Pitic 62 was the major cultivar grown in the Sacramento Valley of California, and by 1974, CDL-6 caused widespread damage throughout the region (Line 1980). CDL-6 was detected in region 1 in 1976 and is now common in all regions but region 7. Druchamp's susceptibility to CDL-6 differentiates CDL-6 from the other previously described races. Two of the commercial cultivars grown in region 6 at that time were Pitic 62 and Produra. CDL-6 produced infection type 8-9 on seedlings of Pitic 62, whereas the older races initially produced infection type 2 on Pitic 62 and within a week produced an infection type 7. Adult plants of Produra were moderately resistant in the field, but the seedlings clearly differentiated CDL-6 from the older races.

CDL-7 was first detected in early spring 1974 in fields of Paha (Suwon 92/Omar) near Walla Walla in southeastern Washington (region 1). By the end of the growing season, CDL-7 had spread into eastern Oregon, throughout eastern Washington and northern Idaho, and as far north as Creston, Canada. The senior author had anticipated that Paha would become vulnerable to stripe rust in region 1 because of Paha's susceptibility to CDL-2. CDL-2 and CDL-7 are both virulent on plants with resistance from Suwon 92. In addition, CDL-7 is virulent on Heines VII, and CDL-2 is virulent on Chinese 166. From 1974 to 1981, CDL-7 was prevalent wherever Paha was grown, except an area in the northwestern corner of region 1. Wheat in that section may escape the development of early stripe rust because snow mold often destroys the rust-infected leaves and reduces the inoculum and because low precipitation limits the rust development. Also, inoculum is seldom introduced into that section because prevailing winds in region 1 are from the southwest. Growers continued to plant Paha in that northwestern section of region 1 for several years. But in the early 1980's, annual precipitation was greater than usual, fall weather was more favorable for rust development, and snow mold was less severe. Consequently, Paha was severely damaged by the rust, and most growers stopped planting the cultivar. CDL-7 continues to occur in region 1, but its prevalence has decreased. CDL-7 has not been detected in any other region.

CDL-8 was first detected on Yamhill in northwestern Washington (region 5). Yamhill was added as a new cultivar to differentiate CDL-8 from CDL-3. Region 5, with its mild winters and low temperatures in spring and summer, is ideal for epidemics of stripe rust, and local epidemics occur there every year. Because wheat prices increased in 1973, wheat production was expanded in region 5. More than 95% of the wheat in region 5 was planted with the newly released cultivar Yamhill. Losses caused by stripe rust exceeded 50% in 1974. Yamhill is a high-yielding, well-adapted cultivar in region 5, and even though stripe rust usually caused losses of more than 20%, farmers continued to grow Yamhill until 1978, when the superior cultivar Stephens was released. It is highly probable that CDL-8 or other races virulent on Yamhill could have been present before 1974. It is possible that CDL-8 and CDL-4 have identical virulences, because they are both virulent on Heines VII and Alba, the parents of Yamhill. CDL-8 was common in regions 1 and 4 in the late 1970's and has been prevalent in region 7 since the mid-1980's.

CDL-9 is similar to CDL-6 but is also virulent on Heines VII. CDL-9 was detected in 1975 near Walla Walla, WA, on Norco, a new wheat cultivar being increased for release. Norco was never released. CDL-9 is highly virulent on Fielder, a new spring wheat cultivar that was grown in 1974 in the field adjacent to Norco. CDL-9 continues to occur in all regions except region 7.

Four closely related races, all virulent on Fielder, were detected near Walla Walla in 1976: CDL-6 (virulent on Druchamp and Produra), CDL-12 (virulent on Paha and Druchamp), CDL-13 (virulent on Paha, Druchamp, and Produra), and CDL-14 (virulent on Produra). Since Produra provided further identification of the races, it was added to the North American differential set. CDL-12 and CDL-13 do not occur in other regions and occur infrequently in region 1. CDL-14 was rare until the mid-1980's, when it became prevalent in region 6.

CDL-10, CDL-15, CDL-16, CDL-17, and CDL-18 were detected in region 5 in 1976 and 1977. CDL-15 was discovered in 1976 on seedlings of Stephens, a new wheat cultivar being considered for release. Stephens was added as a differential cultivar to identify the race. Stephens has adult-plant resistance, which is effective against all races. Even in the presence of CDL-15, yields of Stephens are greater than yields of Yamhill. Therefore, Stephens replaced Yamhill as the preferred cultivar in region 5. Currently, more than 95% of the wheat acreage in region 5 is Stephens, even though stripe rust reduces the annual yield by 15% or more.

CDL-15 is virulent on Druchamp and Stephens, moderately virulent on Heines VII, and avirulent on Yamhill. In contrast, CDL-10, CDL-16, CDL-17, and CDL-18 are all virulent on Heines VII and Yamhill but not on Druchamp and Stephens. In addition, CDL-10 is virulent on Chinese 166, CDL-16 is virulent on Lee, CDL-17 is virulent on Lee and Chinese 166, and CDL-18 is virulent on Moro. Therefore, Lee was subsequently added to the differential list in order to identify CDL-16 and CDL-17. CDL-10, CDL-15, and CDL-17 remain confined to region 5. CDL-16 and CDL-18 currently occur in region 1.

CDL-19 and CDL-20 are similar to CDL-9 and CDL-6, respectively, but are virulent on Stephens. CDL-19 and CDL-20 were detected in region 1 in 1977. Since then, both races have been prevalent in all regions except region 7.

CDL-11, virulent on Lemhi but not on the other differentials, was detected in regions 5 and 6 in 1976. CDL-11 is rare in both regions but more common in region 6. It has not been detected in other regions.

Lemhi was considered to be susceptible to all races of *P. striiformis* worldwide until 1978, when CDL-21 was collected from triticale (wheat × rye) lines near San Luis Obispo, CA (region 6). CDL-21 is virulent on only Chinese 166, grows slowly on seedlings, produces fewer spores, and is a poor competitor in mixtures. CDL-21 is sometimes collected from wheat and/or triticale in region 6 but has never been collected from other regions.

CDL-22 was first collected from Daws in region 1. Before appearance of CDL-22, all races that were virulent on Fielder were also virulent on Druchamp and/or Produra and could be differentiated by use of those cultivars. However, CDL-22 was virulent on Fielder but not on Druchamp and Produra. So Fielder was added to the differential set. Since then, CDL-22 has been prevalent in region 1, and it is currently prevalent in all regions except region 7. Two similar races are CDL-24 and CDL-26, which were detected in 1981 and 1982, respectively. CDL-24 is also virulent on Paha, and CDL-26 is also virulent on Yamhill. CDL-24 has remained in region 1; CDL-26 has also been collected in regions 4 and 5.

CDL-23 and CDL-25 are like CDL-15 and CDL-19, respectively, but are also virulent on Yamhill. CDL-23 and CDL-25 were detected in 1981 in regions where Yamhill and Stephens are both common. CDL-23 was detected in region 5 and, like CDL-15, has remained confined to that region. CDL-25 was first detected in region 4. The following year, it was detected in regions 1 and 5. CDL-25 is common wherever Stephens is grown extensively and is often associated with CDL-19 and CDL-20, which are also virulent on Stephens. CDL-37 (like CDL-25 but virulent on Lee) was collected in 1987 in region 5 but has not been collected in other regions.

From 1983 to 1987, 11 new races (CDL-27, CDL-28, CDL-29, CDL-30, CDL-31, CDL-32, CDL-33, CDL-34, CDL-36, CDL-38, and CDL-39) were detected in region 1. All 11 races have remained confined to region 1. CDL-27 and CDL-33 were detected on Tyee, a newly released club wheat. Since their detection, they have frequently reduced yields of Tyee by 30% or more. Tyee was added to the differential set to separate CDL-27 and CDL-33 from other races. CDL-31 (virulent on Heines VII, Paha, and Lee) and CDL-38 (virulent on Heines VII and Lee) were collected in central Washington from Hatton, a new hard red winter wheat. CDL-31, CDL-38, and CDL-16 (also virulent on Lee) are most frequently collected from Hatton. CDL-28, CDL-29, CDL-30, CDL-34, CDL-36, and CDL-39 are all virulent on Moro but have additional virulence on other cultivars grown in region 1; i.e., they represent reassortments of the virulence genes in region 1.

Prior to 1985, all races virulent on Stephens were also virulent on Druchamp. Then CDL-35 (virulent on Lemhi and Stephens) was detected in region 6 in the same area where CDL-21 was collected. Original cultures were from triticale and wheat. CDL-35 is slower to sporulate on Stephens than are other races that are virulent on Stephens. CDL-35 has not been detected in any other region.

Geographic Distribution of Races

The seven geographic regions of North America (fig. 4) are determined based on geographic barriers, prevailing winds and other weather, crop management, and occurrence and virulence of races of *Puccinia striiformis*. Region 1 has the greatest number of races (fig. 4) and the races with the greatest range of virulence, i.e., virulence on the greatest number of differential cultivars in North America (table 4). Of the 39 races identified in North America, 31 occur in region 1; of those 31, 26 were first detected in the region. Of the five races that were first detected in other regions, CDL-8, CDL-16, and CDL-18 were detected in region 5, CDL-25 was detected in region 4, and CDL-6 was detected in region 6. Based on the characteristics of the regions, it is deduced that CDL-6, CDL-8, CDL-16, and CDL-18 probably evolved independently in region 1 and were not introduced from other regions. CDL-25 could have been introduced from region 4, but all available evidence indicates that it evolved independently in region 1. Because region 1 is bordered on the west and east by mountains and the prevailing winds are from the southwest, the probability of the introduction of spores from other regions is low.

Region 1 is an important wheat-growing region with an environment that is favorable for rust survival in winter and rust development in spring. During the last 30 years, 3 out of every 4 years have been favorable for severe rust epidemics in fields of susceptible cultivars in region 1. In addition, cropping practices favor the survival and development of rust in region 1. All but one of the major classes of wheat are grown in region 1. Soft white winter wheat and soft white spring wheat are grown primarily in the eastern third of the region (which has the region's highest rainfall) and in irrigated fields throughout the region. Hard red winter wheat is grown primarily in the lower-rainfall areas in the central part of the region. Hard red spring wheat is grown in both the low-rainfall areas and under irrigation in the central and western parts of region 1. Club wheats are grown primarily in intermediate-rainfall areas bordered on the west by the hard red winter wheat and on the east by the soft white

Table 4. Virulence and avirulence of races of *Puccinia striiformis* from seven regions of North America on differential wheat cultivars

North American region[1]	Differential wheat cultivar[2]												
	1	2	3	4	5	6	7	8	9	10	11	12	13
1	V[3]	V	V	V	V	V	A[3]	V	V	V	V	V	V
2	V	V	V	V	A	V	A	V	V	V	A	V	A
3	V	A	V	A	A	V	A	V	A	V	A	V	A
4	V	V	V	V	A	V	A	V	V	V	V	V	A
5	V	V	V	V	A	V	A	V	V	V	V	V	A
6	V	V	V	A	A	V	A	V	A	V	A	V	A
7	V	A	V	A	A	A	A	A	V	A	A	A	A

[1]See fig. 4 for map of regions.
[2]1 = Lemhi, 2 = Chinese 166, 3 = Heines VII, 4 = Moro, 5 = Paha, 6 = Druchamp, 7 = Riebesel 47-51, 8 = Produra, 9 = Yamhill, 10 = Stephens, 11 = Lee, 12 = Fielder, 13 = Tyee.
[3]V = virulent, A = avirulent.

winter wheat. Spring durum wheat is grown by a few farmers, usually under contract. Under drier conditions, winter wheat is planted in August and September; under wetter conditions, it is planted in October and November. Spring wheat is planted in March or April. Thus management of these wheats provides a continuous green host for the survival and development of rust in region 1.

The distribution and prevalence of the races of *P. striiformis* in region 1 are primarily related to susceptibility of the cultivars. The races that are virulent on Moro, Paha, Lee, and Tyee have been consistently most prevalent in the low-to-intermediate rainfall areas where susceptible club wheats and hard red winter wheats are grown. Races virulent on Fielder, Druchamp, Produra, and Stephens have been consistently most prevalent in the southern half of region 1, where Stephens is most commonly grown. Races virulent on Fielder but not Druchamp, Produra, and Stephens are prevalent in the northern half of the region, especially where Daws is grown. Races virulent on Lemhi and Heines VII remain common throughout the region. Races virulent on Yamhill are occasionally detected, and races virulent on Chinese 166 are very rare.

Regions 2 and 3 are somewhat isolated from other regions to the west, and generally their winters are usually unfavorable for the survival of stripe rust. Consequently, severe epidemics are less frequent in those regions. All races of *P. striiformis* identified from 1968 to 1987 in regions 2 and 3 were first detected in region 1. Races virulent on Lemhi, Heines VII, Druchamp, Produra, Stephens, and Fielder (table 4) are prevalent in both regions. Virulences on Chinese 166, Moro, and Yamhill are present in region 2 but not region 3. CDL-5 was prevalent in region 2 in the late 1960's and early 1970's, but its prevalence decreased when Crest wheat was no longer grown. CDL-5 or other races that are virulent on Moro have never been detected in region 3 even though many of the hard red winter wheat cultivars in that region are susceptible to them. All data on the races indicate that the races in regions 2 and 3 originated in region 1. When rust is severe in region 1, rust epidemics often occur later in the season in region 2, and the races that are detected are the same as those that occurred earlier in the eastern and northern sections of region 1. Because of the late development of epidemics, spring wheat cultivars, such as Fielder, Fieldwin, and Sterling (all susceptible to the same races), are the most vulnerable to stripe rust, especially in Alberta, Canada. Races that are virulent on Fielder are common in that area.

The geographic barriers, direction of prevailing winds, favorable temperature and precipitation, and virulence of the races of *P. striiformis* all provide evidence that stripe rust races in region 4 probably originate locally and that the introduction of inoculum from other regions, except by humans, seldom (if ever) occurs. Races virulent on Yamhill and Stephens, the major cultivars grown in region 4, are most prevalent in the region. All races in the region are virulent on Lemhi, and virulence on Heines VII, Druchamp, Produra, and Fielder is common. In the early 1970's, experimental plots were inoculated with races virulent on Chinese 166 and Moro, but those races did not spread very far from the inoculation site, probably because there was and is no selective pressure for the virulences.

Because of mountain barriers and prevailing winds, stripe rust is indigenous to region 5 and rust inoculum does not enter from other regions. Further, because of mild winters and cool springs and summers, stripe rust in region 5 has been severe annually for the last 16 years. Eighteen of the thirty-nine races have been detected in region 5, and nine of the races were first detected there. Seven races (CDL-8, CDL-10, CDL-16, CDL-17, CDL-18, CDL-23, CDL-37) are virulent on Yamhill, and three races (CDL-15, CDL-23, CDL-37) are virulent on Stephens. Yamhill and Stephens are the two major cultivars grown in region 5. One race (CDL-11) is virulent on only Lemhi. The most prevalent races have been and currently are those virulent on Heines VII, Yamhill, Druchamp, and Stephens. Races virulent on Yamhill are also virulent on

Heines VII, and races virulent on Stephens are also virulent on Druchamp. Virulence on Druchamp was probably present in the region for years. Druchamp has been grown in the region and was still grown in the 1970's on Lopez Island in region 5. Races virulent on Yamhill were prevalent in the 1970's, when Yamhill made up more than 95% of the wheat grown in region 5. Most farmers in the region changed to Stephens in 1978. Since then, more than 95% of the wheat has been Stephens, and races virulent on Stephens have been most prevalent. Virulence on Heines VII and Druchamp may be explained by their common association with virulence on Yamhill or Stephens. However, the occurrence of some of the other races cannot be explained by the susceptibility of local cultivars to those races. The presence of races that are virulent on Chinese 166, Moro, Lee, Produra, or Fielder is not related to cultivars grown in the region. Region 5 is a center of major rust diversity, and stripe rust has existed there for more than 100 years (Humphrey et al. 1924). Stripe rust also occurs on several species of wild grasses and on cultivated rye, and those hosts may contribute to the diversity of the races. However, predominant rust collections from those species have been identical to collections from adjacent wheat fields in the region.

Region 6, which is also separated from the other regions, has a favorable environment for rust during the growing season (October to June) but a less favorable environment during summer, when wheat and triticale are not grown as crops. However, those hosts are present as volunteer plants in other crops, along the roadsides, and on irrigation dikes (Line 1976). Eleven races have been detected in region 6, of which four were first detected in the region. Virulences on Lemhi, Heines VII, Druchamp, Produra, Stephens, and Fielder are common (table 4), and virulence on Chinese 166 is more common in region 6 than in other regions. CDL-21 and CDL-35 have been collected from only region 6; CDL-11, which occurs only in regions 5 and 6, is most common in region 6. Each of these three races has a narrow range of virulence (table 3). CDL-21 and CDL-35 appear to have a closer association with triticale than to wheat. The rust occurs on various grasses, especially *Hordeum* sp., and these additional hosts may contribute to the uniqueness of the races in region 6.

Region 7 includes all wheat-producing areas east of the Rocky Mountains. The winter weather in the south is favorable for the survival and increase of stripe rust, but the hot spring and summer weather in the south and hot summers in the north often limit the development of severe epidemics. However, stripe rust has become more prevalent in region 7 since 1984, especially in the South-Central States. Virulence of the races is consistent throughout the region, probably because of prevailing winds and the absence of geographic barriers. Isolates collected from that region and northern Mexico during the period before 1985 were all identified as CDL-3. Since then, a closely related race, CDL-8, has been found in region 7. There may be other virulences that cannot be detected by use of the current differentials. However, of the many cultivars from region 7 that have been tested, none have detected virulences that have not been detected by the current differentials.

Evolution and Relationship of Races

Virulence on Chinese 166 (differential 2) has been known in the United States and Canada since the early 1930's (Newton et al. 1933, Bever 1934, Newton and Johnson 1936), but it has never become a major component of the rust population. CDL-1 (virulent on Chinese 166 and Lemhi) was used to inoculate research plots in regions 1, 2, and 4 throughout the 1960's and 1970's, but CDL-1 was never detected more than 40 km (25 miles) from the inoculation sites and was rare more than 8 km (5 miles) from the sites. Only a few collections of CDL-2 (virulent on Chinese 166 and Paha) have been obtained since 1968. CDL-39 (virulent on Chinese 166 and Moro) was not detected until 1987 and has not been collected since then. During the same period, other races virulent on Paha and/or Moro were prevalent throughout region 1 and contributed to severe epidemics. Seven other races virulent on Paha and eight other races virulent on Moro have been more prevalent than CDL-2 or CDL-39. In region 5, CDL-1 and CDL-10 (virulent on Chinese 166, Heines VII, and Yamhill) were detected in 1976, and CDL-17 (like CDL-10 but also virulent on Lee) was detected in 1977. All three races were rare in the 1970's and were not collected in the 1980's, even though a special effort was made to examine Chinese 166 in the trap plots and to collect specimens from highly susceptible plants. Virulence on Chinese 166 is most common in region 6, where CDL-1 and CDL-21 (virulent on only Chinese 166) were detected in 1978. CDL-21 has been collected in region 6 every year since then but is not always prevalent.

North American races that are virulent on Chinese 166 are not aggressive on wheat and do not appear to be well adapted to that host. CDL-1, CDL-17, and CDL-21 in the greenhouse on seedlings are slow to sporulate and produce fewer spores than do other races. When CDL-1 was used extensively to inoculate experimental plots, by the end of the growing season other naturally occurring races became the predominant races in the plots. CDL-21 was first collected from triticale in southwestern region 6, where stripe rust commonly occurs on wild *Hordeum* sp. (Line 1976). Also, isozyme analyses indicate that CDL-21 differs from other North American races that were tested (personal communication with A.P. Roelfs, Cereal Rust Laboratory, St. Paul, MN). Because races virulent on Chinese 166 have been associated with triticale in region 6, rye may have contributed to the occurrence of races virulent on Chinese

166 in region 5 during the 1970's. At that time, moderately susceptible rye was occasionally grown as a green manure crop. That cropping practice is now uncommon.

Virulence on Lemhi and Heines VII occurs throughout North America (table 4). CDL-21 is the only race that does not attack Lemhi, and 26 of the 39 races that have been identified are virulent on Heines VII (table 3). After races that attack Lemhi, races with virulence on Heines VII are the most prevalent and occur most often with other virulences (table 5). All collections of races that are virulent on Yamhill or Lee are also virulent on Heines VIII. CDL-3 (virulent on Lemhi and Heines VII) was the most prevalent race in regions 1, 2, 3, and 4 from the early 1960's to the mid-1970's and in region 6 from the 1960's to 1973. CDL-3 continues to be collected in those regions and is prevalent in region 7. All data on the relationships and evolution of the races indicate that CDL-3 is an ancestor of the most prevalent and well-adapted races of *P. striiformis* in North America. Historically, there may have been some selectivity for CDL-3. The spring cultivar Marfed, which is resistant to other older races but susceptible to CDL-3, was grown in region 1 during the 1960's. The major winter wheat cultivars were susceptible to all races, but Marfed may have provided a late season "bridge" for the summer survival of CDL-3. In regions 4 and 5, Yamhill (which has resistance from Heines VII) has eliminated races that are avirulent on Heines VII. Triumph hard red winter wheat, once widely grown in Oklahoma and Kansas, appears to have the same resistance as Heines VII and may have provided selective pressure for CDL-3 and CDL-8 in region 7. Most cultivars currently grown in North America do not have the resistance of Heines VII, but virulence on Heines VII continues to be common.

Table 5. Number of races of *Puccinia striiformis* that show dual association[1]

Differential wheat cultivar		Differential wheat cultivar number												
Name	Number	1	2	3	4	5	6	7	8	9	10	11	12	13
Lemhi	1		4	26	9	8	11	0	9	11	7	5	18	2
Chinese 166	2	4		2	1	1	0	0	0	2	0	1	0	0
Heines VII	3	26	2		6	5	6	0	4	11	5	5	12	2
Moro	4	9	0	6		2	1	0	1	1	0	0	4	0
Paha	5	8	1	3	2		0	0	0	0	0	1	2	0
Druchamp	6	11	0	6	1	0		0	8	3	6	0	9	0
Riebesel 47-51	7	0	0	0	0	0	0		0	0	0	0	0	0
Produra	8	9	0	4	1	0	8	0		2	4	0	9	0
Yamhill	9	10	2	11	1	0	3	0	2		2	3	5	1
Stephens	10	7	0	5	0	0	6	0	4	2		0	4	0
Lee	11	5	1	5	0	1	0	0	0	3	0		0	0
Fielder	12	18	0	12	4	2	9	0	9	5	4	0		0
Tyee	13	2	0	0	0	0	0	0	0	1	0	0	2	
Total dual associations		109	10	80	25	19	44	0	37	41	28	15	65	5

[1]Number of races virulent on two different cultivars.

Eighteen of the thirty-nine races identified in North America are virulent on Fielder, and virulence on Fielder frequently occurs in combination with other virulences (table 5). The virulence that is detected with Fielder has probably existed in North America for at least 60 years. One of the two races reported in the early 1930's (Newton et al. 1933, Bever 1934, Newton and Johnson 1936) was virulent on Heines Kolben. The resistance of Fielder is probably identical or similar to that of Heines Kolben. All North American races that are virulent on Fielder are also virulent on Heines Kolben, and all races not virulent on Fielder are not virulent on Heines Kolben. Races that are virulent on Fielder can be put into two population groups: Group A (CDL-6, CDL-9, CDL-12, CDL-13, CDL-19, CDL-20, CDL-25, and CDL-37) and Group B (CDL-22, CDL-24, CDL-26, CDL-27, CDL-28, CDL-30, CDL-33, CDL-34, and CDL-36). Group A races are also virulent on Druchamp and Produra; group B races are not virulent on Druchamp or Produra but are virulent on Heines VII. Many of the races in group B are also virulent on the club wheats. Following the detection of CDL-6 in region 6 in 1973 and CDL-9 in region 1 in 1975, races virulent on Fielder have been prevalent in all regions except region 7. Group A races have been more prevalent than group B races in all of the regions except the northern half of region 1 during the 1980's. The distribution of the two groups can be explained, at least partially, by selective virulence for local cultivars. Group B is often collected from Daws and the club wheats in the northern half of region 1. Group A has been more commonly associated with the regions where Stephens wheat is grown. Four of the eight races in group A are also virulent on Stephens. It is not clear why the virulence of group A on Druchamp and Produra is associated with virulence on Fielder (table 3). Only two races (CDL-15 and CDL-23) have been collected that are virulent on Druchamp and not Fielder, and those two races remain confined to region 5. These results indicate that there could be some common genes for resistance in those cultivars, especially Produra and Fielder.

Virulence on Yamhill has been most prevalent in regions 4 and 5, where Yamhill was commonly grown in the 1970's. However, the virulence occurs in other regions where Yamhill is not grown, especially in region 7. Based on the pedigrees of cultivars grown in region 7, the resistance of some of the cultivars is probably the same as the resistance of Yamhill.

Virulence on Lee was first detected in region 5 in 1977. Until 1983, races virulent on Lee were collected from only region 5, usually late in the growing season on spring wheat cultivars in the trap plots. Several of the spring wheat cultivars are susceptible to those races that are virulent on Lee. In 1983, CDL-31 (virulent on Lemhi, Heines VII, Paha, and Lee) was obtained from a single field of Hatton hard red winter wheat in central region 1. Since then, CDL-31 and CDL-38 (virulent on Lemhi, Heines VII, and Lee) have been more frequently collected, usually from Hatton. Hatton has been consistently more susceptible to the races that attack Lee. One of the parents in Hatton is a spring wheat line that may have some common resistance with Lee.

Races virulent on Moro, Paha, and Tyee are primarily found in region 1, probably because of the selective pressure provided by those club wheats. The races have been most prevalent where those susceptible cultivars are grown.

Most of the races with new virulences were detected after the release of new cultivars with high resistance to stripe rust: race CDL-5 on Moro in 1968; CDL-6 on Pitic 62 in 1973; CDL-7 on Paha in 1974; CDL-8 on Yamhill in 1974; CDL-9 on Norco and Fielder in 1975; CDL-15, CDL-19, and CDL-20 on Stephens in 1976 and 1977; CDL-22 on Daws in 1980; CDL-27 on Tyee in 1983; and CDL-31 on Hatton in 1983. When released, each of the cultivars was highly resistant to the races of *P. striiformis* in the region, but new races that could circumvent their resistance became prevalent within 3 years.

Some cultivars are susceptible in the seedling stage but resistant in the adult stage at relatively high tempertures. This resistance is known as high-temperature, adult-plant (HTAP) resistance (Qayoum and Line 1985). HTAP resistance shows no race specificity and is more effective as plants become older and temperatures increase during the growing season. HTAP resistance remains durable even though seedlings are susceptible. The high seedling resistance of Daws and Stephens was quickly circumvented by new races, but the HTAP resistance of Daws and Stephens has remained durable. Because of its durability, HTAP resistance is preferred in a cultivar, but HTAP resistance has not always been easy to incorporate into cultivars (such as the club wheats) that may have specific agronomic and quality requirements. The race-specific resistance of the club wheats Moro, Barbee, Faro, Jacmar, Paha, and Tyee has been effective for only a short time.

During the 20 years of these studies, no conclusive evidence was seen for the classically interpreted "stabilizing selection." Within those years, less virulent races did not always become more prevalent when the race-selective cultivars were no longer grown. In addition, as discussed earlier, some of the least virulent races (such as CDL-1, CDL-11, CDL-21, CDL-35) were also less aggressive. They grew very slowly and required a longer time to sporulate. However, in general, the first races to appear with new virulences have a relatively narrow range of virulence; that is, the new races are virulent on only a few of the differential cultivars. This is true even though the predominant races that were present in a region or area at the time of their detection had more virulences. This suggests that it is easier for new virulences to arise when there are fewer virulence genes or that races with fewer virulence genes are more aggressive; i.e., the races have better survival ability or increase faster. Therefore, they more quickly become a predominant component of the population. Most of the new virulences have been closely associated with virulence on Heines VII and/or Fielder; i.e., the new virulences have been races that attack Heines VII and/or Fielder (table 3).

Within a few years after the detection of new virulences, reassortment of those virulences appears to occur. In this way, races with various new combinations of virulence became common. It is not known how the new races evolved, but based on what is known about the cereal rusts and the life cycle of *P. striiformis*, certain inferences can be made. *P. striiformis* has no known host for completion of its sexual cycle. If such a host does exist, monitoring and epidemiological evidence indicate that the host does not occur in the wheat-producing regions of North America where stripe rust occurs. Therefore, any new virulences or new combinations of virulences must occur in asexual cells. We assume that new genes for virulence have evolved by mutation, but we do not know when the mutation occurred. If the new genes were present in the population before release of the resistant cultivars, they must have been at a very low level, because reevaluation of earlier collections shows no virulence on the new differential cultivars. This is best documented for region 1. After new virulent races are detected, many new combinations of virulence are detected. Under controlled conditions in a greenhouse, new races are very stable. New races from mixtures of races under those conditions have never been detected. Studies with other rusts suggest that new races may have evolved by additional mutations, nuclear exchange, heterokaryosis, or some type of parasexual system. However, there are no conclusive studies on the mechanisms of recombination in *P. striiformis*.

After the appearance of new races with their relatively narrow range of virulence, the races become more complex and the races with a greater number of virulences become more common (table 6). Thus several of the most recently identified races are the most virulent races (table 3). This trend cannot be explained by the selectivity of combined resistance genes in cultivars, as occurs in stem rust. The breeding strategy for control of stripe rust in North America is aimed at combining HTAP resistance or new sources of specific resistance with other desirable characteristics to obtain high yielding cultivars. There is no effort to "pyramid" genes into a single cultivar. However, the diversity of resistance in cultivars grown within a region may provide the same selective pressure.

Expression of Resistance and Possible Genes for Resistance in Differentials

Expression of resistance differs among the differential cultivars. Resistance is expressed as an infection type 2 in Lemhi, Chinese 166, Moro, Druchamp, Produra, Stephens, Lee, Fielder, and Tyee. Susceptibility in those cultivars is expressed as infection type 8 or 9, depending on environmental conditions. Expression of resistance is similar in Heines VII except in response to CDL-15 and CDL-23. Virulence of CDL-15 and CDL-23 initially appears as infection types 2-3, but sporulation increases as the pathogen continues to grow on the leaf, ultimately producing infection types 6-7. Resistance in Riebesel 47-51 is expressed as a zero or infection type 1, depending on environmental conditions. A low percentage of the plants of Chinese 166, Druchamp, and Stephens are susceptible to all races. The off-type plants have been present in all tested sources, and attempts to remove the susceptible plants from the seed sources have been unsuccessful. A small percentage of the plants of Fielder and Yamhill produce intermediate infection types instead of low types and are not used in the evaluation.

Table 6. Examples of evolution of *Puccinia striiformis* from relatively simple races to more complex races with new combinations of virulences

Year detected	CDL[1] race	Virulent on differential wheat cultivar[2]	Total differentials[3]
		Common virulence on Lemhi and Chinese 166:	
1960	1	1,2	2
1963	2	1,2,5	3
1976	10	1,2,3,9	4
1977	17	1,2,3,9,11	5
		Common virulence on Lemhi and Moro:	
1968	5	1,3,4	3
1977	18	1,3,4,9	4
1983	28	1,3,4,12	4
1983	29	1,3,4,5	4
1983	30	1,4,6,8,12	5
1984	34	1,3,4,5,12	5
1985	36	1,3,4,9,12	5

Table 6—Continued

Year detected	CDL[1] race	Virulent on differential wheat cultivar[2]	Total no. differentials[3]
		Common virulence on Lemhi, Heines VII, and Yamhill:	
1974	8	1,3,9	3
1976	10	1,2,3,9	4
1977	16	1,3,9,11	4
1977	17	1,2,3,9,11	5
1977	18	1,3,4,9	4
1981	23	1,3,6,9,10	5
1981	25	1,3,6,8,9,10,12	7
1982	26	1,3,9,12	4
1984	33	1,3,9,12,13	5
1985	36	1,3,4,9,12	5
1987	37	1,3,6,8,9,10,11,12	8

[1]Cereal Disease Laboratory (CDL) type race.
[2]1 = Lemhi, 2 = Chinese 166, 3 = Heines VII, 4 = Moro, 5 = Paha, 6 = Druchamp, 7 = Riebesel 47-51, 8 = Produra, 9 = Yamhill, 10 = Stephens, 11 = Lee, 12 = Fielder, 12 = Tyee.
[3]Total number of differentials on which race is virulent.

Differentials with known specific genes would be highly useful, but information on the genes for resistance in the stripe rust differentials is either unavailable or inadequate. However, some hypotheses can be made regarding genes for stripe rust resistance in the North American differentials, based on the response of cultivars to various races, on the parentage of differentials, and on comparison of cultivar-race interactions. Chinese 166, Heines VII, Moro, Riebesel 47-51, and Lee have been reported to have genes Yr_1, Yr_2, Yr_{10}, Yr_9, and Yr_7, respectively (Stubbs 1985). Resistance in Fielder appears to be the same as resistance in Heines Kolben, which has been reported to be Yr_6 (Stubbs 1985). Yamhill appears to have Yr_2 from Heines VII and one or more genes from Alba. The remaining North American differentials each have at least one unique gene for resistance.

Lemhi was initially included in the differential set because it is susceptible to all races known worldwide at that time. The discovery of CDL-21 showed that Lemhi has a gene for resistance and that virulence for that gene is highly prevalent throughout North America. Paha and CI013749 (sister cultivars) differentiate the same races and are interchangeable. Stephens, which has Yr_{3a} and/or Yr_{4b} based on its Nord Deprez parent, may have a gene that is the same as a gene in Druchamp but must have at least a second gene that is different. Before the discovery of CDL-35, all races that were virulent on Stephens were also virulent on Druchamp. However, the virulence of CDL-35 on Stephens is different from the virulences of CDL-15, CDL-19, CDL-20, CDL-25, and CDL-37 on Stephens. CDL-35 is slow to sporulate, produces few spores, and produces a slightly lower infection type (infection type 7) than do the other races. This suggests that if Druchamp and Stephens both have the same gene(s), Druchamp must have an additional gene(s). In North America, stripe rust has not been detected on cultivars with genes Yr_5 or Yr_8, and none of the differentials have genes Yr_5 or Yr_8.

Based on these interpretations, data on the resistance of major cultivars in North America, and historical data, it can be stated that virulence for Yr_1 and Yr_6 has existed for at least 60 years in North America. Virulence for Yr_1 was not prevalent during 1960-88 and does not appear to be well adapted to the major commercial cultivars grown in North America, which apparently do not have Yr_1. Virulences for the gene(s) in Lemhi and for Yr_2 in Heines VII are prevalent throughout North America. Virulences for Yr_6 in Fielder and for the gene(s) in Druchamp, Produra, and Stephens are prevalent in the western regions. Virulence for the additional gene(s) in Yamhill occurs in regions 1, 2, 4, 5, and 7 but is most prevalent in regions 4, 5, and 7. Virulence for Yr_7 in Lee is present in regions 1, 4, and 5 but has not been prevalent during the 20 years of monitoring. Virulence for Yr_{10} in Moro has been detected in regions 1, 2, 4, and 5; has been prevalent in region 2; and remains prevalent in region 1. Virulences for the gene(s) in Paha and Tyee have been detected in only region 1, where they are prevalent. Virulences for Yr_5, Yr_8, and Yr_9 have not been detected in any of the regions.

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